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2:1 Atrioventricular Block During Atrioventricular Node Reentrant Tachycardia

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Objectives. **The purpose of this study was to determine the incidence and to clarify the mechanism of 2:1 atrioventricular (AV) block during AV node reentrant tachycardia induced in the electrophysiology laboratory.**

Background. **In patients with 2:1 AV block during AV node reentrant tachycardia, the absence of a His bundle potential in the blocked beats has been considered evidence of intranodal, lower common pathway block.**

Methods. **In consecutive patients with AV node reentrant tachycardia, the incidence of 2:1 AV block and the response to atropine and a single ventricular extrastimulus was observed.**

Results. **Persistent 2:1 AV block occurred in 13 of 139 patients with AV node reentrant tachycardia. A His bundle deflection was present in the blocked beats in eight patients and absent in five. Patients with 2:1 AV block had a shorter tachycardia cycle length than did patients without such block (mean ± SD 312 ± 32 vs. 353 ± 55 ms, $p < 0.01$). Atropine did not alter the 2:1 block in any patient. In every patient, a single ventricular extrastimulus introduced during the tachycardia converted the 2:1 block to 1:1 conduction.**

Conclusions. **The incidence of induced 2:1 AV block during AV node reentrant tachycardia is ~10%. The lack of a response to atropine and the consistent conversion of 2:1 block to 1:1 conduction by a ventricular extrastimulus indicate that, regardless of the presence or absence of a His bundle potential in blocked beats, 2:1 block during AV node reentrant tachycardia is due to functional infranodal block.**

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Although 2:1 atrioventricular (AV) block during AV node reentrant tachycardia is known to occur (1–3), the incidence and the site of the 2:1 block have not been well characterized. In previous studies, the absence of a His bundle depolarization in the blocked beats was considered evidence of intranodal, lower common pathway block or block occurring between the AV node and the His bundle (1–3). The purpose of this prospective study was to determine the incidence and mechanism of 2:1 AV block during AV node reentrant tachycardia in the electrophysiology laboratory.

Methods

Patient characteristics. The subjects of this study were 139 consecutive patients who underwent an electrophysiology procedure and were found to have inducible AV node reentrant tachycardia. There were 45 men and 94 women with a mean age ± SD of 45 ± 16 years. The mean duration of symptoms was 14 ± 11 years. The mean cycle length of the tachycardia was 350 ± 54 ms. The mean His-ventricle interval during sinus rhythm was 45 ± 8 ms. Ten of the 139 patients had a prolonged His-ventricle interval (range 60 to 75 ms, mean 64 ± 6), but no patient had pathologic infranodal block during rapid atrial pacing. Six patients had coronary artery disease; the remaining 133 patients had no structural heart disease.

Electrophysiologic testing. Electrophysiology tests were performed after informed consent was obtained and after discontinuation of all antiarrhythmic agents for at least 5 half-lives. Three quadripolar electrode catheters with 2-5-2–mm interelectrode spacing were inserted into a femoral vein and positioned in the high right atrium, His bundle position and right ventricular apex. Whenever necessary, one of these catheters was manipulated into the coronary sinus. Several electrocardiographic leads and the intracardiac electrograms were displayed on an oscilloscope and recorded at a paper speed of 100 mm/s on a Mingograph 7 recorder (Siemens-Elema, Solna, Sweden). The His bundle electrogram was recorded at a gain setting of 80 mm/mV. Pacing was performed with a programmable stimulator (Bloom) using stimuli at twice the diastolic threshold and 2 ms in duration. The conduction properties and refractory periods of the AV node were determined. Previously described criteria were used to establish the diagnosis of AV node reentrant tachycardia (4).

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Table 1. Baseline Electrophysiologic Characteristics in Patients With 1:1 and 2:1 Atrioventricular Conduction During Atrioventricular Node Reentrant Tachycardia

	Conduction		
	1:1 (n = 126)	2:1 (n = 13)	p Value
Age (yr)	45 ± 15	42 ± 20	0.43
Male/female ratio	39:81	4:8	0.11
Sinus CL (ms)	803 ± 153	793 ± 151	0.71
AH interval (ms)	85 ± 23	77 ± 18	0.39
HV interval (ms)	46 ± 8	41 ± 4	0.09
AV block CL (ms)	362 ± 94	327 ± 65	0.16
VA block CL (ms)	352 ± 82	283 ± 53	0.004
Fast pathway ERP	335 ± 97	287 ± 57	0.11
Slow pathway ERP	276 ± 60	292 ± 52	0.97
AVNRT cycle length	353 ± 55	312 ± 32	0.04

Values expressed as mean ± 1 SD. AH = atrial-His; AVNRT = atrioventricular node reentrant tachycardia; CL = cycle length; ERP = effective refractory period; HV = His-ventricle; VA = ventriculoatrial.

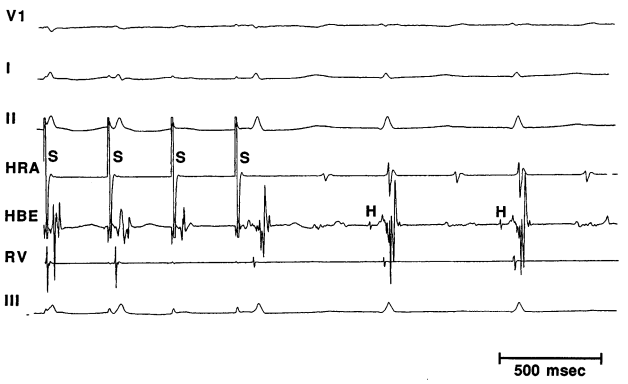


Figure 1. Example of AV node reentrant tachycardia with 2:1 AV block induced by atrial overdrive pacing at a cycle length of 270 ms. A "long-short" sequence is set up by the third from last atrial stimulus, which results in AV node block, and the second from last atrial stimulus, which conducts to the ventricle. This condition predisposes to functional block in the His bundle. A His bundle potential is absent in the blocked beats. H = His bundle potential; HBE = His bundle electrogram; HRA = high right atrium; RV = right ventricle; S = stimulus.

Study protocol. Sustained 2:1 AV block was defined as 2:1 AV block that persisted during tachycardia until the introduction of an intervention. If 2:1 AV block was present during AV node reentrant tachycardia, the catheter used to record the His bundle electrogram was manipulated to maximize the amplitude of the His potential in conducted and blocked beats. During sustained 2:1 AV block, the diastolic interval was scanned with a single ventricular extrastimulus. In addition, 1 mg of atropine was injected intravenously during tachycardia when there was sustained 2:1 AV block.

Statistical analysis. Values are expressed as mean ± 1SD. Continuous variables were compared using a paired *t* test. A p value <0.05 was considered significant.

Results

Incidence and method of induction of sustained 2:1 AV block (Table 1). Thirteen of 139 patients had reproducibly inducible sustained 2:1 AV block during AV node reentrant tachycardia. Sustained 2:1 AV block was induced twice in 3 patients, and three or more times in 10. The episodes of AV node reentrant tachycardia in which there was sustained 2:1 AV block were induced by programmed stimulation with a single atrial extrastimulus in six patients, atrial overdrive pacing in five and ventricular overdrive pacing in two. The method of induction was reproducible in 12 of 13 patients. In each patient, 2:1 AV block was present at the onset of induced episodes of AV node reentrant tachycardia (Fig. 1).

Differences between patients with and without 2:1 AV block (Table 1). The 13 patients in whom 2:1 AV block during AV node reentrant tachycardia was reproducibly inducible had a mean tachycardia cycle length of 312 ± 32 ms and a mean ventriculoatrial block cycle length of 283 ± 53 ms. These values were significantly shorter than the mean tachycardia cycle length of 353 ± 55 ms ($p < 0.05$) and the mean ventriculoatrial block cycle length of 352 ± 82 ms ($p < 0.01$) in the 126 patients who did not have reproducibly inducible 2:1 AV block during AV node reentrant tachycardia.

The patients with and without reproducibly inducible 2:1 AV block during AV node reentrant tachycardia, did not differ significantly in age, gender, incidence of structural heart disease, sinus cycle length, baseline atrial-His or His-ventricle interval, AV block cycle length or the effective refractory period of the fast or slow pathway.

His bundle potential during 2:1 AV block (Table 2). The His-ventricle interval during sinus rhythm and during tachycardia was normal in each patient who had 2:1 AV block during AV node reentrant tachycardia. A His bundle potential was present in blocked beats during 2:1 AV block in eight

Table 2. His-Ventricle Interval and His Potential Amplitude in Patients With 2:1 Atrioventricular Block During Atrioventricular Node Reentrant Tachycardia

Pt. No.	HV (ms)	His Potential Amplitude (mV)	
		Conducted Beats	Blocked Beats
1	40	0.46	0.46
2	45	0.13	0.01
3	40	0.38	0
4	40	0.24	0.05
5	45	0.31	0.04
6	35	0.15	0
7	35	0.30	0
8	40	0.38	0.04
9	35	0.06	0.06
10	40	0.10	0
11	50	0.04	0
12	35	0.05	0.01
13	45	0.20	0.01

HV = His-ventricle interval during sinus rhythm and tachycardia; Pt = patient.

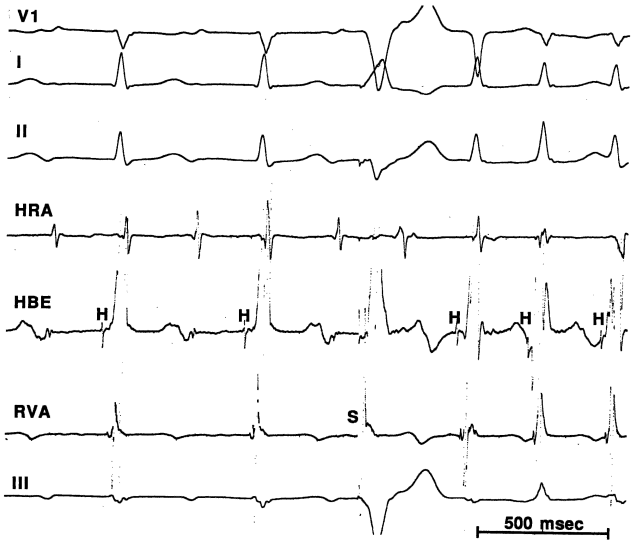


Figure 2. Example of 2:1 AV block during AV node reentrant tachycardia and conversion to 1:1 conduction by a ventricular extrastimulus. The tachycardia cycle length varies from 230 to 280 ms. A His bundle potential is absent in the blocked beats. RVA = right ventricular apex; other abbreviations as in Figure 1.

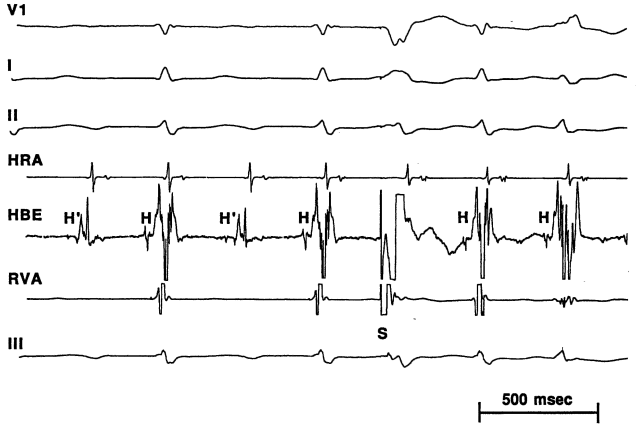


Figure 3. Example of 2:1 AV block during AV node reentrant tachycardia and conversion to 1:1 conduction in response to a ventricular extrastimulus. There is a small His bundle potential with an amplitude of 0.01 mV in the blocked beats, in contrast to a His bundle potential of 0.05 mV in the conducted beats. The ventricular extrastimulus is coincident with the His bundle potential. H′ = His bundle potential in the blocked beats; other abbreviations as in Figures 1 and 2.

patients and absent in five. Among the eight patients in whom a His bundle potential was present in the blocked beats, the maximal amplitude of the His bundle potential ranged from 0.01 to 0.46 mV (mean 0.05 ± 0.13 mV) in these beats in contrast to 0.04 to 0.46 mV in conducted beats (mean 0.22 ± 0.14 mV, $p < 0.001$).

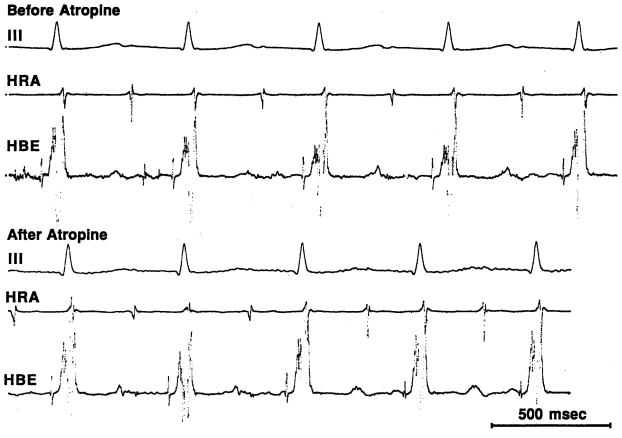
Response to premature ventricular depolarizations. In every patient, a single ventricular extrastimulus introduced during AV node reentrant tachycardia converted the 2:1 AV block to 1:1 conduction (Fig. 2). The ratio of the coupling interval of the ventricular extrastimulus to the RR cycle during AV node reentrant tachycardia with 2:1 AV block ranged from 0.77 to 0.89. In two patients, the ventricular extrastimulus responsible for the conversion to 1:1 AV conduction occurred coincident with the His bundle potential (Fig. 3).

Response to atropine. Atropine was administered in 10 patients during AV node reentrant tachycardia with 2:1 AV block. A significant decrease in the atrial cycle length during tachycardia was observed after the administration of atropine (286 ± 33 ms vs. 256 ± 31 ms, $p < 0.01$). Atropine had no effect on the AV block in any patient (Fig. 4).

Discussion

Main findings. The results of this study demonstrate that the incidence of reproducible, sustained 2:1 AV block during induced episodes of AV node reentrant tachycardia is ~10%. A His bundle potential is absent in blocked beats in ~40% of patients who have 2:1 AV block, and in the remaining patients, the His bundle potential may range from being rudimentary to large in amplitude. However, irrespective of whether or not a His bundle potential is present in blocked beats, the AV block persists after the administration of atropine, suggesting that the site of block is not in the AV node. In addition, a ventricular extrastimulus introduced during the 2:1 AV block consistently results in 1:1 conduction, indicating that the AV block is functional and that the level of block is infranodal whether or not a His bundle potential is present during AV block. Therefore, these results demonstrate that what has been previously thought to be 2:1 AV node block in the lower common pathway of the AV node reentrant tachycardia circuit is more likely to be intra-Hisian block.

Figure 4. Example of 2:1 AV block during AV node reentrant tachycardia and the response to 1 mg of atropine administered intravenously. The **top panel** demonstrates a baseline tachycardia cycle length of 290 to 300 ms and the absence of a His bundle potential in the blocked beats. The **bottom panel** demonstrates that the tachycardia cycle length shortens to 250 to 260 ms after atropine administration but that the 2:1 block persists. Abbreviations as in Figure 1.



Factors predisposing to 2:1 AV block during AV node reentrant tachycardia. Three findings in this study suggest that the 2:1 AV block that occurred during episodes of AV node reentrant tachycardia was not caused by pathologic AV block. 1) The phenomenon of 2:1 AV block during AV node reentrant tachycardia was independent of demographic variables and was not associated with His-ventricle interval prolongation or pathologic infranodal block during atrial pacing. 2) The modes of initiation of episodes of AV node reentrant tachycardia associated with 2:1 AV block usually were compatible with exposure of the His bundle to a relatively long cycle length followed by a relatively short cycle length, consistent with functional block. 3) ≥11 patients who had episodes of AV node reentrant tachycardia with 2:1 AV block also had several episodes of AV node reentrant tachycardia at the same cycle length associated with 1:1 AV conduction. These findings all indicate that the 2:1 AV block during AV node reentrant tachycardia occurred on a functional basis and was not related to a pathologic abnormality in AV conduction.

The tachycardia cycle length and the ventriculoatrial block cycle length were significantly shorter in patients who had episodes of 2:1 AV block than in patients who did not. This finding suggests that a more rapid input into the bundle of His after a relatively long cycle length that prolongs refractoriness in the His bundle predisposed to the occurrence of 2:1 AV block in the patients in this study.

Response to atropine. The vagolytic effect of atropine would be expected to shorten refractoriness and to improve conduction in the AV node but not in the bundle of His (5,6). In this study, the persistence of 2:1 AV block during AV node reentrant tachycardia in all of the patients who received atropine suggests that the level of block was below the AV node. The significant shortening of the tachycardia cycle length after the administration of atropine indicates that the 1-mg dosage used was sufficient to exert a clinically apparent vagolytic effect.

Response to a ventricular extrastimulus. In every patient in this study who had sustained 2:1 AV block during AV node reentrant tachycardia, an appropriately timed ventricular extrastimulus consistently converted the 2:1 AV block to a 1:1 AV relation. This observation provides additional evidence that the 2:1 AV relation was caused by functional as opposed to pathologic block.

Resolution of the AV block with a ventricular depolarization also indicates that the block was occurring in the His-Purkinje system. The response to pacing implies that the ventricular extrastimulus resulted in retrograde depolarization of the bundle of His, eliminating the "long-short" sequence that was responsible for perpetuation of the 2:1 block. A pattern of 2:1 block in the AV node would not be expected to resolve with retrograde penetration of the node by a ventricular depolarization, because the perpetuation of AV node block does not depend on a "long-short" sequence. Furthermore, in two patients, the ventricular depolarization which converted the 2:1 block to a 1:1 AV relation occurred when the His bundle was refractory, ruling out any possibility that the AV block was occurring in the AV node.

His bundle potential. The presence or absence of a His bundle potential in blocked beats usually is helpful in determining whether the block is occurring within or below the AV node. In the present study, ~40% of patients with 2:1 block during AV node reentrant tachycardia had no discernible His bundle potential in the blocked beats, an observation that might lead to the conclusion that the AV block was arising in the AV node. However, the 2:1 AV block always resolved with a ventricular depolarization, and never resolved after the administration of atropine, whether or not a His bundle potential was present in blocked beats. These findings provide strong evidence that the level of AV block always was below the AV node, even when a His bundle potential was not present in the blocked beats.

The absence of a His bundle potential in blocked beats in some patients and the marked variability in the amplitude of the His bundle potential in the other patients who had 2:1 AV block during AV node reentrant tachycardia may be explained by variable degrees of penetration of the His bundle. Block in the proximal portion of the His bundle, by resulting in the depolarization of only a small portion of the His bundle, might explain the absence of a detectable His bundle potential or a His bundle potential that is much smaller than in conducted beats. In contrast, block in the distal portion of the His bundle could result in a His bundle potential that had an amplitude similar to that of the His bundle potential of conducted beats.

Previous studies. Previous reports have indicated that 2:1 block during AV node reentrant tachycardia occurs within the AV node, between the AV node and the bundle of His or in the proximal His bundle. Schmitt et al. (1) described a patient with 2:1 AV block during AV node reentrant tachycardia and concluded that the absence of a His bundle potential in blocked beats was most likely due to block in the lower common pathway of the AV node reentrant tachycardia circuit. Miles et al. (2) described a single patient with 2:1 AV block during AV node reentrant tachycardia in whom a His bundle potential was absent in blocked beats; they suggested that the site of AV block was in the proximal His bundle or distal AV node. Wellens et al. (3) demonstrated that 2:1 AV block during AV node reentrant tachycardia occurred in 13% of 67 patients, which is similar to the 10% incidence found in the present study. In the study by Wellens et al. (3) six of nine patients had infranodal block, based on the presence of a His bundle potential in the blocked beats. In three of the nine patients, no His bundle potential was recorded in blocked beats, and the site of block in these patients was thought to be between the AV node and the His bundle. In each of these previous studies, conclusions about the level of AV block were based only on the presence or absence of a His bundle potential in the blocked beats and not on the response of the AV block to physiologic maneuvers. The results of the present study demonstrate that the response of 2:1 AV block during AV node reentrant tachycardia to maneuvers such as atropine

administration or ventricular pacing is uniform and always consistent with intra-Hisian block, regardless of the status of the His bundle potential in the blocked beats.

The results of the present study do not preclude the possibility of AV node block during AV node reentrant tachycardia. For example, DiMarco et al. (7) described a patient who had AV block during AV node reentrant tachycardia and presented strong evidence that the block was occurring in the AV node. However, that patient had a Wenckebach pattern of AV block during AV node reentrant tachycardia, whereas all patients in the present study had a pattern of 2:1 AV block.

Limitations. A limitation of this study is that the incidence of 2:1 AV block during AV node reentrant tachycardia of ~10% found in this study may apply only to episodes of AV node reentrant tachycardia induced in the electrophysiology laboratory. Because variables such as autonomic tone and the mode of induction may be different for spontaneous as opposed to induced episodes of AV node reentrant tachycardia, the incidence of 2:1 AV block during spontaneous episodes of AV node reentrant tachycardia remains unclear.

A second potential limitation is that catheter movement during 2:1 AV block may account for the changes observed in the His bundle recording. The lack of a His bundle potential in the blocked beats may be a result of a more distal His bundle recording. However, such a potential limitation would suggest the presence of a His potential in the blocked beats and this would further support the contention that the level of block is occurring infranodally.

Conclusions. Our results demonstrate that the absence of a His bundle potential in blocked beats during sustained 2:1 block in the setting of AV node reentrant tachycardia is not a reliable indicator of block within the AV node. The mode of initiation and the responses to atropine and ventricular pacing all indicate that sustained 2:1 AV block during AV node reentrant tachycardia is caused by functional block within the His bundle, whether or not a His bundle potential is present in blocked beats.

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